NOTES
ON
OPHTHALMOLOGY.

SYLLABUS AND MEMORANDA FOR THE USE OF STUDENTS.

BY

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PREFACE.

THESE Notes were written for the use of students attending my Course on Ophthalmology, and are a synopsis of these Lectures.

It is hoped that the student will be able to follow the Lecture with greater facility and to better advantage if he have read a summary of it beforehand.

It is only by the use of a syllabus of this nature that a Lecturer can attempt to deal with such an extensive subject as ophthalmic surgery in the time apportioned to it in the curriculum.

I wish, however, to emphasize the fact that Ophthalmology can only be learned in the Out-patient room and wards. Lectures and Text-books are only valuable in so far as they supplement clinical teaching.

In the compilation of these Notes I have frequently consulted various standard works, and notably those of my predecessor, Professor Priestley Smith, to whom I am also indebted for kindly criticism and friendly suggestions.

J. JAMESON EVANS.

Scheme for the Examination of Cases

ANAMNESIS: Heredity. Personal history. Past and present illnesses. Habits. Occupation.

GENERAL OBSERVATION.

Note general aspect of patient, especially face, orbit, brows, scalp, head posture, &c.

LOCAL PHYSICAL EXAMINATION.

EYELIDS: Position, movements, margins, size of palpebral fissures. Condition and position of cilia. Method of everting upper and lower lids.

LACHRYMAL APPARATUS: Note swelling in region of sac or gland. Pressure over sac for regurgitation of contents. Note position and size of puncta. Syringe.

EYEBALLS: Position, direction, size, proptosis or recession. Movements: determine muscle or muscles paralysed. Squint: Determine its character. Measure amount or angle of squint.

Latent squint: Test for muscle-balance. Maddox Rod and tangent scales; perimeter, &c.

Tests for binocular vision.

CONJUNCTIVA: Palpebral [illegible] caruncle. Method of inspecting fornices. Note readiness [illegible] Discrimination between conjunctival [illegible] Discharge: Note [illegible] Examine bacteriologically.

CORNEA: [illegible] Note position, character and age of opacities, [illegible] or vessels. Use focal light, oblique illumination, transmitted light, loupe, microscope.

Surface: [illegible] or irregularities. Note distortion by reflection of candle (test) or Placido's disc. Breach of surface. Note fluorescein. Sensation. Cotton wool, blast or current of air.

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EYEBALLS: Position, direction, size, proptosis or recession. Movements: determine muscle or muscles paralysed. Squint: Determine its character. Measure amount or angle of squint.

Latent squint: Test for muscle balance. Maddox Rod and tangent scales; exclusion test.

Tests for binocular vision.

CONJUNCTIVA: Palpebral, bulbar, retrotarsal; plica and caruncle. Method of exposing fornices. Note vascularity, thickness, and surface. Distinguish between conjunctival, episcleral and ciliary congestions. Discharge: Note its character and amount. Examine bacteriologically.

CORNEA: Size, transparency, surface, sensation.

Note position, character and age of opacities, infiltration or vessels. Use daylight, oblique illumination transmitted light, loupe, loupe-mirror.

Surface: Note loss of polish or irregularities. Note distortion of reflection of window bars or Placido's disc.

Breach of surface: Note reflections, use fluorescin.

Sensation: Cotton wool, hair, current of air.

SCLEROTIC: Surface, colour, thickening or thinning. Distinguish between solid and cystic bulging. Trans-illumination.

ANTERIOR CHAMBER: Depth, contents. Loupe.

IRIS: Colour, lustre, pattern, plane of surface, tremor, coloboma, adhesions (anterior or posterior), embryonic remains. Compare two eyes carefully. Loupe.

PUPILS: Size, shape, reactions—direct (light) associated (accommodation and convergence) and consensual. Test in daylight and in dark room, and compare two eyes carefully.

LENS: Note colour of pupil, dots, streaks, or diffuse grey haze. Confirm by focal illumination and transmitted light. Position and character of opacities. Loupe-mirror. Note position and stability of lens. Tremor of iris.

VITREOUS: Note if clear. Opacities—Loupe-mirror, and diffuse light. Hæmorrhages and gross opacities by focal illumination or diffuse transmitted light.

TENSION: Fingers. Tonometer.

FIELD OF VISION: Perimetry—Hand. Perimeter. Campimeter. Tests for colour scotoma.

OPHTHALMOSCOPIC EXAMINATION: Direct. Indirect.

DETERMINATION OF VISUAL ACUITY: Form, colour, and light sense. Objective and subjective methods with and without Mydriatic. Accommodation.

GENERAL SYSTEMIC EXAMINATION.

Nervous system, kidneys, heart, blood-pressure, etc. Nose and Accessory Sinuses.

DISEASES OF THE EYELIDS.

STRUCTURE AND FUNCTIONS OF THE EYELIDS.

MALFORMATIONS: Coloboma, Epicanthus, Cryptophthalmos, Lagophthalmos.

PTOSIS or DROOPING OF THE UPPER LID: Congenital, Paralytic (third and sympathetic), Traumatic, Cicatricial, Senile, Mechanical, Hysterical.

Treatment: Principles of operative correction.

BLEPHARITIS CILIARIS: Pustular, Seborrhœic, Secondary (to conjunctivitis or nasal infection).

Causes: Constitutional, Staphylococcus, Errors of Refraction.

Complications: Ectropion, Epiphora, Trichiasis, Entropion.

Treatment: Deal with cause. Antiseptic cleanliness, removal of scabs, antiseptic ointments (Mercurial, Zinc, Ichthyol, Lead, etc.). Vaccines.

HORDEOLUM or STYE: Suppuration in the follicle of an Eyelash, due to staphylococcal infection.

Treatment: Epilate Eyelash, hot fomentations, incise. Constitutional treatment, antisepsis and vaccines as for Blepharitis.

CHALAZION, Meibomian or Tarsal Cyst: Adenitis and periadenitis.

Cause (?) Staphylococcus (?) Xerosis Bacillus.

Diagnosis: from Hordeolum.

Treatment: Incision from inside, vertical; occasionally from outside, horizontal. Scoop out contents, excise sac if thick. Local antisepsis.

ENTROPION, or inversion of the edge of the lid, generally associated with Trichiasis, or inward misdirection of the eyelashes, which leads to irritation and congestion of the eyeball and ulceration of the cornea.

Causes: i. Cicatricial.—Injuries, burns, trachoma, blepharitis.

ii. Spasmodic.—In children with phlyctenular ophthalmia.

In old people with lax tissues (senile), after operations.

iii. Congenital.

Treatment: Remove cause where possible. Temporary eversion by sticking-plaster or collodion. Epilation or electrolysis for Trichiasis. Operative: Excision of elliptical piece of musculocutaneous tissue for senile and congenital and mild cicatricial; Excision of wedge-shaped groove of tarsus, or splitting and shifting of lid margin for advanced cicatricial entropion. Van Milligen's operation. Canthoplasty.

ECTROPION, or eversion of the lid margin.

Causes: i. Chronic conjunctivitis and blepharitis.

ii. Senile relaxation and paralysis of orbicularis (viith).

iii. Cicatrisation after burns, wounds, caries of orbital margin.

Treatment: Correct cause, attend to conjunctiva and tear-passage. Advanced cases: Excision of hypertrophied tissue, Snellen's sutures, plastic operations, Tarsorrhaphy.

SYMBLEPHARON: Partial or complete adhesion of eyelid to eyeball, due to inflammation and apposition of raw surfaces.

Treatment: Prophylactic, plastic operation.

ANKYLOBLEPHARON: Adherence of lids to each other at their margins.

BLEPHAROPHIMOSIS: Small palpebral aperture.

LAGOPHTHALMOS, or inability to close lids completely.

Causes: Paralysis viith. Spasms of levator or of Muller's muscle. Cicatrisation from injuries, etc. Proptosis:—Graves's Disease, orbital growths, or cellulitis. Congenital shortness or absence (ablepharia) of lids.

BLEPHAROSPASM: Spasmodic contraction of orbicularis palpebrarum, which may be clonic, tonic, or fibrillary, and is a manifestation of accompanying disease or neurosis.

Causes: Reflex (diseases and injuries of cornea, conjunctiva, fissure of lids). Errors of refraction. Neuroses (hysteria, senility).

MISCELLANEOUS DISEASES OF LIDS.

Sypilis, Herpes ophthalmicus, Vaccine Blepharitis, Distichiasis.

Rodent ulcer, Dermoid, Angioma.

INJURIES OF THE LIDS: Contusions, incisions, burns, etc. Characterised by œdema and subcutaneous ecchymosis in consequence of loose texture of tissues. Vertical wounds gape, horizontal do not owing to disposition of fibres of orbicularis. Ecchymosis of lower lid in fracture of base of skull. Emphysema of lids in fracture of orbital walls opening into nasal sinuses.

Treatment: General principles. Careful apposition of lid margin when cut; intermarginal suture.

DISEASES OF THE CONJUNCTIVA.

CONJUNCTIVA:

Structure. Sub-divisions. Blood supply. Functions. Methods of examination.

CONJUNCTIVITIS:

Causes: i. Physico-Chemical:

Injuries, foreign bodies, dust, smoke, pollen, strong light (especially ultra-violet rays), heat, wind, errors of refraction, etc.

Chemical agents: Atropine, pilocarpine, silver salts, mercury and iodine, gases and foul air, etc.

Toxins: Exanthemata, lithæmia, alcoholism, metastatic gonorrhœa, tuberculo-toxæmia—(? Phlyctenular conjunctivitis).

ii. Infective:

- Mucopurulent
  - Koch-Weeks' Bacillus—acute, epidemic.
  - Pneumococcus—acute or chronic.
  - Morax-Axenfeld Diplo-bacillus—chronic blepharo-conjunctivitis.
  - Staphylococcus (?) Phlyctenular.
- Purulent - Gonococcus.
- Membranous -
  - Streptococcus.
  - Klebs-Loeffler's Bacillus.
  - Pneumococcus.
- Granular -
  - Tubercle Bacillus.
  - Trachoma (?) organism.
  - Parinaud's conjunctivitis.

MUCOPURULENT, or CATARRHAL CONJUNCTIVITIS.

Acute, subacute, or chronic: Chronic conjunctivitis may follow an acute attack, or arise from chemico-physical causes, or be due to certain infective organisms (*q.v.*).

Symptoms: Vary in degree rather than character. Stiffness, dryness, burning, itching, sensations of F.B. photophobia, lachrymation, coloured rings round lights, Gumming of lids by discharge. Lids swollen and red in acute, margins only in chronic. Conjunctiva: red, swollen and rough, occasionally sub-conjunctival hæmorrhages. In chronic forms, follicular thickening. Discharge: Mucopurulent and abundant, or mucoid, or watery, foamy or flocculent (soap), fibrinous.

Complications: Marginal corneal ulcers (diplo-bacillary), Iritis (pneumococcal), phlyctenules (Koch-Weeks'), Blepharitis, Epiphora, cold in the head.

Treatment: Prophylactic measures to prevent spread. Remove cause. Locally: Cleansing with antiseptic lotions—boric, mercurial, etc. Instillations of antiseptic and astringent drops:—Protargol 5%. Argyrol 10 %. Zinc sulphate $\frac{1}{10}$ % to 2 % (diplo-bacillary). Ointments along lid margins:—boric, mercurial, zinc, ichthyol (1 %). Avoid bandages. Vaccines in intractable cases.

PHLYCTENULAR CONJUNCTIVITIS. Scrofulous or Eczematous Conjunctivitis. Small papules or pustules on bulbar conjunctiva, generally near limbus surrounded by circumscribed area of congestion. Often multiple, relapsing and recurrent, break down and form small ulcers.

Complications: Extension into cornea (P. Keratitis), nasal catarrh, impetigo or eczema of face, glandular tuberculosis, digestive disturbances.

Treatment: Constitutional. Climate. Diet. Tonics. Mild aperients and intestinal antiseptics. Local: Mild antiseptic lotions—Protargol drops (1 %). Calomel dusting. Ung. H.O.F. (1 %).

GONORRHŒAL CONJUNCTIVITIS: Purulent ophthalmia.

(a) In the new-born (Ophthalmia Neonatorum).
(b) In the adult.

(a) Ophthalmia Neonatorum. Cause of 25 %—30 % of blindness in institutions.

Causes: Gonococcus in 60 %—70 % of cases. Other organisms of infective conjunctivitis and bacterium coli responsible for the rest. Infection generally occurs *after birth*, occasionally during delivery, seldom before.

Symptoms: Begin 1—3 days after infection. Both eyes generally affected. Lids: red, œdematous. Discharge: serous, later creamy and yellow. Conjunctiva: congested, chemosed, ecchymosed, later thickened, velvety vascular granulations.

Complications: Corneal infiltration, ulceration, perforation, Iritis, anterior polar cataract, leucoma adherens, staphyloma, panophthalmitis, systemic infection.
Later, Nystagmus.

Course: Lasts 2—6 weeks often leaving chronic conjunctivitis.

Treatment: (i) Notification; (ii) Prevention; (iii) Curative.

(ii) Prevention. Treat Gonorrhœa in the mother. Cleanse baby's lids before they are opened, if possible, and its hands and arms as soon as they appear. Always wash the face afterwards in fresh and separate water. Cleanse eyelids again after bath, and instil 1 % silver nitrate, or Protargol 10 %, Argyrol 20 %, Sophol 5 %. Surgeon, midwife, and mother to adopt surgical cleanliness to prevent secondary infections. Inspect the eyes daily, *carefully observe the cornea, but do not touch it.*

(iii) Curative. Frequent and thorough cleansing of the conjunctival sac with mild, warm lotion, *e.g.*, saline, boric, oxycyanide of mercury (1·4000), perchloride of mercury (1·10000), potass per-

manganate (1·5000), peroxide of hydrogen (1·3). Instillation of drops of Argyrol, Protargol, or Sophol. Smear edges of lids with non-irritating ointment—boric (2 %), peroxide of hydrogen (1:3), Iodol (5 %). Painting of everted lids with 2 % silver nitrate *by the surgeon*, in cases with granular thickening of the conjunctiva. Atropine ($\frac{1}{2}$ %) in cases with corneal ulcer and iritis. Zinc drops for late chronic conjunctivitis. Attend to the general health of baby and mother.

(b) Gonorrhœal Conjunctivitis in the Adult: Infection from urethra or other eye, by hands, towels, etc., often affects one eye only.

Symptoms: As (a), but chemosis much more pronounced, and consequently disastrous corneal complications of frequent occurrence. Malaise and feverishness.

Treatment:

(i) Prophylaxis. Cleanliness and care in cases of urethritis. Protection of healthy eye with Buller's shield.

(ii) Curative: As Ophthalmia Neonatorum. Vaccines and serum.

Metastatic Gonorrheal Conjunctivitis: A much milder form of conjunctivitfs, with little discharge or pain; secondary to urethral gonorrhea (*c.f.* gonorrheal "rheumatism"), but not due to direct infection. The gonococcus is not found in the conjunctival discharge.

MEMBRANOUS CONJUNCTIVITIS. Generally Diphtheritic, may be Streptococcal, Gonococcal, Pneumococcal, etc.

DIPHTHERITIC: Lids brawny and stiff; submembranous surface grey; other evidences of Diphtheria.

NON-DIPHTHERITIC: Lids soft and easily everted; submembranous surface red and vascular.

Stages: (i) Membranous; (ii) Blenorrheal; (iii) Cicatricial.

Complications: Necrosis of cornea and conjunctiva, Symblepharon.

Treatment: Isolation. Buller's shield. Antitoxin and vaccines. Local: As in purulent ophthalmia, substituting quinine for silver nitrate in membanous stage.

TRACHOMA, or GRANULAR CONJUNCTIVITIS. Contagious; epidemic in certain countries and among certain races. (?) Cause.

Pathology: Conjunctivitis, with papillary outgrowths, and the formation of greyish translucent "sago-grain" granulations showing the structure of lymphoid follicles.

Complications: Mixed infection (catarrhal, gonorrhœal, etc.), Pannus (superficial vascular Keratitis), corneal ulcers generally along margin of pannus, Trachomatous Ptosis, Entropion, Trichiasis, Ectropion, Xerosis.

Treatment: Prophylactic. Isolation Local: Copper sulphate, Silver nitrate, perchloride of mercury, jequirity, X-rays, radium, carbon dioxide snow. Expression. Excision.

TUBERCULOSIS OF THE CONJUNCTIVA.

PARINAUD'S CONJUNCTIVITIS.

SPRING CATARRH.

PINGUECULA, PTERYGIUM, XEROSIS.

TUMOURS OF THE CONJUNCTIVA. Cysts, lipoma, lymphangietasis, papilloma, epithelioma and sarcoma, etc.

INJURIES OF THE CONJUNCTIVA. Foreign bodies, generally lodged in subtarsal ridge, wounds and burns —lime, acids, alkalies, hot metals, etc.

CORNEA AND SCLERA.

STRUCTURE AND FUNCTIONS. Structures perforating sclerotic.

DISEASES OF THE CORNEA.

KERATITIS: Inflammation of the Cornea.

(i) Suppurative Keratitis:

(a) Ordinary simple corneal ulcer—from injury, foreign body, etc.

(b) Corneal ulcer, secondary to conjunctivitis.

(c) Ulcer serpens. Hypopyon ulcer. Pneumococcal. Lachrymal infection. Streptococcus, etc.

(d) Desiccation Keratitis, with exophthalmos, or lagophthalmos.

(e) Keratitis neuroparalytica (v^{th}).

(f) Phlyctenular and fascicular ulcers (*v.* conjunctivitis).

(g) Chronic serpiginous, or rodent ulcer (Mooren).

(h) Marasmic ulcer. Keratomalacia.

(ii) Non-Suppurative Keratitis:

(a) Superficial:

(i) Pannus.

(ii) Vesicular. Herpetic. Bullous. Recurrent erosion.

(b) Deep:

(i) Interstitial.

(ii) Sclerosing Keratitis.

(iii) Associated with Uveal disease.

Corneal Ulcer.

Symptoms: Photophobia, lachrymation, grittiness, pain more or less severe, defective vision when pupillary area involved.

Signs: Loss of substance in the cornea, varying in extent, depth, and shape. Conjunctival and ciliary injection.

(ii) Non-Suppurative Keratitis.

(*a*) *Superficial.*

(i) Pannus : *v.* Trachoma.

(ii) Vesicular Keratitis :

(a) Herpes Corneæ : Recurrent eruption of small vesicles on the surface of the cornea, lasting for few hours and accompanied by pain and irritation, which pass off when vesicles rupture.

(b) Herpes Zoster Ophthalmicus, often associated with iritis and large vesicle formation on cornea.

(c) Herpes febrilis : Febrile attack ; small corneal vesicles, which rupture and form dendritic (branching) ulcer. (? Mycotic.)

(d) Keratitis Bullosa : Large bullæ in old leucomatous or glaucomatous eyes.

(e) Recurrent erosion of cornea : Epithelium repeatedly detached in large bullæ after injury by blunt objects—finger nail, garden stake, etc.

(*b*) *Deep.*

Interstitial Keratitis : Diffuse parenchymatous Keratitis. Chronic inflammation of the corneal parenchyma associated with deep-seated opacities and ciliary injection.

Causes : Congenital syphilis—onset sixth to twentieth year of life. Acquired syphilis (rare). Tubercle.

Symptoms : Pain (slight), photophobia, impaired vision. Greyish infiltration of cornea generally starting at periphery and spreading towards centre ; often becomes mottled (yellow-white) or infiltrated by deep-seated vessels (salmon patch). Surface of cornea steamy. Little tendency to ulceration, but weakened cornea may give way (myopia or staphyloma). Systemic signs of congenital syphilis, teeth, ears, nose, head, etc

Course: Both eyes usually affected, either simultaneously or successively. Duration 2 to 12 months. Opacities though dense have remarkable capacity for clearing up, but close examination with loupe-mirror will often demonstrate empty vessels in cornea. Relapses not infrequent, and these or original trouble may be started by minor corneal injuries.

Complications: Uveal tract generally affected (iritis, choroiditis, cyclitis, etc.), as shown by K.P., staphyloma, etc. Choroiditis indicates syphilis rather than tubercle. Latter also generally shows nodules on iris, and gives tuberculin but not Wasserman reaction. Keratitis of acquired syphilis is generally unilateral.

Treatment: Constitutional. Cod liver oil, tonics, and fresh air often better than Hg and KI in congenital syphilis. Salvarsan no use. Tuberculin valuable in T. Keratitis. Local: Atropine. Hot fomentations. Leeching in acute stage. Shade from light. Late (subsiding) stages—Ung. H.O.F., with or without massage; subconjunctival injections—NaCl, $Hg(CN)_2$, Dionin.

Sclerosing Keratitis: Scleritis associated with extension of inflammation into cornea, producing an opaque infiltration of the corneal tissue.

Treatment: v. Scleritis.

Keratitis Profunda, with uveal disease. An opacity is produced in the deeper layers of the cornea about its centre. It is probably due to diffusion of toxins from the anterior chamber. It is especially associated with cyclitis—septic, syphilitic, tubercular, or auto-toxæmic.

Treatment: As for Irido-cyclitis. Treat infective or toxic focus.

CONICAL CORNEA, or Kerato-Conus. Consists in a gradual thinning and bulging of the cornea into a conical form under normal intra-ocular pressure. The process generally begins soon after puberty, but may do so at any period of life. Progress is slow and may become stationary. There are no signs of inflammation.

Symptoms: Gradual failure of vision. Conical shape of cornea evident on its examination, alteration of reflexes (window, Placido's disc, etc.). Myopic astigmatism. Grey opacity at apex, which is generally eccentric.

Treatment: Correction of refraction by glasses. Operative: Excision of apex of cone. Electro-cautery followed if necessary by optical iridectomy.

INJURIES OF CORNEA: Burns, F.B.'s, perforating wounds. All injuries should be treated with every antiseptic precaution, and sources of infection, especially lachrymal sac, treated as well.

Foreign Bodies imbedded in cornea removed with grooved or triangular needle.

Perforating Wounds: Special treatment for complications, *e.g.*, prolapse of iris, cataract. Bed. Atropine. Operation.

Burns: Those by chemicals—acids, alkalies, lime—should be treated in bed, permanent effects being much more severe than would appear from primary signs. Use physiological antidote solutions. For lime burns, remove all particles of lime, wash with olive oil, and instil syrup of cane sugar. Prevent symblepharon with oils or ointments, probe or shell.

TUMOURS OF THE CORNEA: Dermoid, Myxoma, Polypus, Epithelioma, Sarcoma.

DISEASES OF THE SCLERA.

SCLERITIS AND EPISCLERITIS.

A localised inflammation characterised by the presence of more or less elevated congested and discoloured patches in the sclera (scleritis), or episcleral tissues (episcleritis).

Causes: (i) Tubercle.
(ii) Syphilis (Gummatous).
(iii) Toxæmia—tubercle, gonorrhœa, autotoxæmia, osteo-arthritis, rheumatism, accessory nasal sinus disease.

Symptoms: Pain, photophobia, lachrymation, tenderness on pressure, phlyctenlike bright red or violet spot not far from cornea (episcleritis), patch more swollen or bulging and blue, with signs of implication of uvea and cornea (scleritis), and may be followed by permanent pigmentation of affected area, staphyloma, myopia, or secondary glaucoma.

Treatment: Constitutional to combat cause; tuberculin and anti-tuberculous regime. Mercury, iodides, salicylates. Surgical treatment for nasal disease. Local: Atropine. Hot fomentations. Leeches. Subconjunctival injections. Massage with Ung. H.O.F.

STAPHYLOMA, or ECTASIA. (a) Partial. (b) Total.

A bulging of the sclera not due to thickening.

Sites: Ciliary, equatorial, posterior pole.

Causes: (i) Inherent weakness.
(ii) Secondary to scleral or uveal inflammation.
(iii) Prolonged increased intraocular tension.
(iv) Buphthalmos (total) *v.* congenital glaucoma.

Diagnosis: From solid growths; by history, tension, ophthalmoscope, and transillumination.

Treatment: Iridectomy for ciliary and equatorial, with increased tension. If advanced and painful, excision or evisceration of globe.

INJURIES OF THE SCLERA.

Wounds and Rupture. Injury of other coats. Escape of vitreous or lens (expulsion or subconjunctival dislocation). Prolapse of parts of uvea. Danger of sympathetic ophthalmia, intraocular hæmorrhage. F.B. in globe.

Treatment: Antiseptics. Sutures if gaping. Atropine. Excision if danger of sympathetic disease, persistent inflammation and minus tension. Retained F.B. to be extracted if possible. Forms of electro-magnets and their uses.

UVEAL TRACT.

(i) Iris. (ii) Ciliary Body. (iii) Choroid.

Structure, Functions. Vascular and nervous supplies.

DISEASES OF THE IRIS.

Congenital Anomalies: Persistent pupillary membrane, Coloboma, Aniridia, Polycoria, Heterochromia.

Pupillary Disturbances: Mydriasis, Miosis, Argyll-Robertson pupil, Hippus, Anisocoria, Corectopia.

Iritis and Iridocyclitis:

Classification:

(i) Plastic.
(ii) Serous or Serofibrinous. } Acute or chronic.
(iii) Purulent—traumatic, secondary, or metastatic.
(iv) Hæmorrhagic.
(v) Granulomatous—tubercle, node.

Ætiology:

(i) Infective—syphilis, tubercle, trauma, infectious diseases.
(ii) Toxæmic—Gonorrhea, oral sepsis, rheumatism and gout, diabetes, auto-toxæmia, infectious diseases.
(iii) Secondary—to diseases of cornea, sclera, uvea.
(iv) Sympathetic.

Symptoms and signs:

Ocular or periorbital pain; photophobia; lachrymation; impaired vision; constitutional disturbance—furred tongue, malaise, &c.; œdema of lids; circumcorneal injection; ciliary tenderness; turbidity and other modifications of aqueous; deposits on the posterior surface of the cornea (K.P.); change in colour and obscuration of

markings of iris; impaired mobility of iris; miosis or irregularity of pupil; exudation into pupil; synechiæ posterior.

Complications:

(i) Occlusion and seclusion of pupil by exudate. Hypopyon, Hyphaema.

(ii) Iris bombé and secondary glaucoma.

(iii) Cataract.

(iv) Extension of inflammation to contiguous structures. Panophthalmitis.

Treatment:

Rest—to eyes and body: shade from light; Constitutional and local remedies for causal factors:—iodides, mercury, salicylates, tuberculin, quinine, aperients and intestinal antiseptics, &c. Local: dry or moist heat; atropine (caution in serous irido-cyclitis with + tension); leeches to temple; iridectomy for synechiæ posterior and secondary glaucoma; paracentesis for increased tension in serous cyclitis.

Sympathetic Ophthalmia (Chronic plastic iridocyclitis or uveitis).

Healthy eye becomes the seat of a destructive inflammation transferred from the other eye, which has been the subject of a similar inflammation—persistent or recurrent—usually as the result of a perforating injury.

The injured eye = exciting eye.

The other eye = sympathising eye.

Ætiology: Ciliary wounds, especially in weakly children. F.B. in globe. Cataract extraction with prolapse of iris. Perforating corneal ulcer with incarceration of iris. Malignant tumours of the uvea.

Incubation period: Onset, generally about three weeks after original injury. Extreme limits two weeks to 20 years or more (with recurrent inflammation). After prophylactic excision, seven weeks quarantine.

Symptoms and signs: Photophobia; lachrymation; asthenopia; subacute conjunctival and ciliary congestion, with more or less pain and ciliary tenderness; turbid aqueous or K.P.; synechiæ posterior; occlusio pupillæ, minus tension; vitreous opacities; sometimes neuroretinitis. Lymphocytosis. Its course is generally chronic with subacute exacerbations, and is apt to be followed by cataract, detached retina, &c.

Histologically: Lymphocyte infiltration with epithelioid and giant-cell formations.

Mode of transmission: Not definitely ascertained. (?) Spread of germs or toxins by way of optic nerve or lymphatics in its sheath.

Treatment: Prophylactic antiseptic treatment of wounds and ulcers. Mercury, sod. salicylate, Salvarsan. Excision of injured eye when useless, and fellow eye sound or showing signs of inflammation; if injured eye not useless and fellow eye already affected, don't excise exciting eye. If fellow eye sound and injured eye not useless, weigh risks and let patient share responsibility of retention. Excision of injured eye after sympathetic inflammation is well established does not materially modify its course.

Injuries of the Iris:

Iridodialysis, Mydriasis, Retroflexion, Irideremia.

Tumours of the Iris.

Sarcoma, Melanoma, Cysts (congenital and traumatic).

Choroid & Retina

Really any structure, choroid a blood bath for retina. Composed of 3 rows of [illegible], largest [illegible] outside, smallest nearest retina. In front of choroid [illegible] is pigment epithelium & rods & cones. In front of these is a layer of [illegible] cells, then layer of ganglion cells, axons of which pass to optic nerve. On internal side there are branches of central artery of retina — occasionally anastomoses of these with choroidal vessels. Cells of retina correspond to ganglion cells of [illegible]. [illegible] together at macula, from there there is a [illegible] to the periphery where only rods are present. Colour vision & all acuity of (vision) (about [illegible]) is due to cones but peripheral portion [illegible] [illegible] in dim light. Takes 20 to 30 mins for eyes to become dark adapted.

DISEASES OF THE CHOROID.

CONGENITAL DEFECTS: Coloboma, Albinism.

CHOROIDITIS:

(i) Suppurative: Trauma, infective or septic embolus (metastatic).

(ii) Non-suppurative, or Exudative. Syphilis, tubercle, toxæmia.

(i) SUPPURATIVE CHOROIDITIS: Acute panophthalmitis. Lids œdematous and red, aqueous and cornea cloudy; often Hypopyon. Acute pain, except in metastatic form. In subacute form yellow fibro-plastic mass in vitreous (pseudoglioma), which may result in phthisis bulbi.

Treatment: Evisceration of globe for acute suppurative panophthalmitis. Excision or evisceration for shrunken globe.

(ii) NON-SUPPURATIVE.

Symptoms: Failure of vision, owing to involvement of macular area, or the presence of opacities in the vitreous. Muscæ, photopsia, metamorphopsia or scotomata, secondary iridocyclitis, cataract or optic atrophy. Ophthalmoscopically: Scattered (disseminated) or single ("solitary patch") foci, soft yellowish, milky or greyish red, organising to white atrophic areas, with pigmented borders. Secondary pigmentary deposits in retina. Tubercular choroiditis may be miliary or solitary.

Treatment: Constitutional, for syphilis, tubercle, etc. Atropine, if cyclitis present. Shade from light. Blood-letting (?). Subconjunctival injections.

DEGENERATIVE CHANGES IN THE CHOROID.

(i) Central degeneration and stretching in myopia.

(ii) Central senile guttate spots.

(iii) Colloid degeneration of lamina vitrea.

These are associated with depreciation of vision, owing to interference with the nutrition of the overlying retina.

TUMOURS OF THE CHOROID.

Sarcoma. Generally obviously pigmented (melanotic), occasionally pigment less obvious (leuco-sarcoma). Generally seen in second half of life. Four stages recognised: (i) intraocular quiescent; (ii) glaucomatous; (iii) perforating; (iv) metastatic.

Ophthalmoscopically: More or less rounded and vascular mass, detaching retina directly, with or without secondary subretinal effusion.

Transillumination: diminished pupillary illumination.

Treatment: Excision of globe, in stages i. and ii. Exenteration of orbit, in stage iii. Palliative, stage iv. Expectation of life after stage iii., about two years.

Carcinoma. Secondary to carcinoma elsewhere.

Treatment: Nil.

INJURIES. Wounds. Ruptures by contusion appear as crescentic tears concentric with optic disc.

RETINA. OPTIC NERVE. CHIASMA. TRACTS. CENTRES.

Anatomy and histology of retina. Special areas. Ophthalmoscopic appearances. Blood supply. Lymph circulation. Structure of optic nerve and tracts. Course of visual fibres from retina to cortical centres.

DISEASES OF THE RETINA.

CONGENITAL ANOMALIES: Opaque nerve fibres, coloboma, night blindness, colour blindness.

RETINITIS.

(i) Infective: Trauma, pyæmia, syphilis.

(ii) Toxæmic: Albuminuria, diabetes, arteriosclerosis, blood diseases.

(iii) Secondary: Neuro-retinitis, choroido-retinitis, hyalitis.

(iv) Phototraumatic: Sun, snow, electric or other intense light.

(v) Degenerative: Retinitis pigmentosa, albescens, etc.

Symptoms and signs: Impaired vision, scotoma, photopsia, metamorphopsia. Dilatation and tortuosity of veins, tortuosity, kinking, lining, and thickening (bright reflex) of arteries (arteriosclerosis), hæmorrhages—flame-like (albuminuria and blood diseases) or punctate (diabetes and arteriosclerosis). Exudations shown by œdema or cloudiness (syphilis and acute nephritis), white spots diffused or arranged in star figure at Y.S., (chronic nephritis), or in circle round macula (R. circinata). Blurring of the optic disc, and even papill-œdema (acute albuminuria). Prognosis: Visual. Vital.

Treatment: Constitutional for primary disease. (?) Premature labour in albuminuric retinitis of pregnancy. Rest and shade. Protective smoked glasses against phototraumatism.

RETINITIS PIGMENTOSA: Pigmentary degeneration of the retina.

Causes: Heredity, consanguinity, deafmutism, (?) syphilis. Characterised by a prolonged course beginning in youth. Gradual failure of vision, marked in subdued light—night-blindness; peripheral contraction of the visual field or ring scotoma; deposit of bonecorpuscle-shaped masses of pigment in the retina; contraction of retinal vessels; atrophy of optic nerve; choroidal changes, posterior polar cataract.

OBSTRUCTION OF RETINAL VESSELS.

(1) Thrombosis of vein.
(2) Thrombosis and embolism of artery.

(1) Thrombosis of Central Vein. Generally at or behind laminal cribrosa.
(*a*) From vascular sclerosis.
(*b*) From neuro-retinitis.
May be followed by canalisation of clot, secondary thrombosis in artery or glaucoma.

Symptoms: Sudden loss of vision, varying in degree according to site of obstruction.

Ophthalmoscopically: O.D. swollen and edges obscured. Marked engorgment and tortuosity of veins. Extensive flame-shaped retinal hæmorrhages and white degenerative or exudative patches. Occasional recovery occurs; apt to result in permanent loss of vision from recurrent hæmorrhages or glaucoma.

Treatment: Shade from light; blood-letting; constitutional treatment of causative disease.

(2) Thrombosis and Embolism of Artery. Retinal arteries are end arteries. Block may occur at disc or nearer periphery.

Causes: Endocarditis—Embolism.
Endarteritis —Thrombosis.

Symptoms: Sudden failure of vision in one eye, with (thrombosis) or without (embolism) premonitory transient failures. Ophthalmoscopic appearances characteristic during the first 24 hours or so, viz.: white œdema of posterior pole of the eye, with cherry-red spot at the macula. Arteries thread-like or beaded. Occasional marginal hæmorrhages. Atrophy of optic disc follows. Central vision may be preserved by cilio-retinal artery. If branch only affected, signs localised and symptoms restricted.

Treatment: Massage, paracentesis, vasodilators.

RETINAL HÆMORRHAGES: May arise from

(1) Retinitis—albuminuric, diabetic, &c.
(2) Blood disorders—leukæmia, purpura, &c.
(3) Vascular Sclerosis.
(4) Glaucoma.
(5) Injuries.
(6) Myopia with distension of globe.

Treatment: Constitutional. Always examine urine. Avoid congestion of head, straining and stooping.

DETACHMENT OF THE RETINA. Separation of the retina from the choroid and pigment epithelium.

Causes: (1) Blows.
(2) High myopia.
(3) New growths—generally choroidal.
(4) Shrinking of vitreous.
(5) Hæmorrhage or serous exudation from choroid.

Symptoms: Impaired vision; distortion and unsteadiness of images, muscæ. The detached portion of the retina is thrown forward (use + lens), is greyish red or white, often uneven and showing wavy movements; the retinal vessels are bent, tortuous, and abnormally dark and lose their light reflex; tension often minus.

Optic atrophy

[illegible] disc – [illegible] – [illegible] white
[illegible]
[illegible] of [illegible] [illegible] found in cases of [illegible]
[illegible]
[illegible] of optic [illegible] – [illegible] primary [illegible]
[illegible] & [illegible]
[illegible] degeneration of [illegible]
[illegible]
vessels very small – optic atrophy (standing) – also found in [illegible]
[illegible] optic atrophy [illegible] general [illegible]
– Tabes, G.P.I. disseminated sclerosis – [illegible]
Disc white [illegible] small [illegible]
inflammatory changes – [illegible]
[illegible] field [illegible] of the [illegible]
take their ordinary course

[illegible] chiasma [illegible]
[illegible] pituitary. [illegible]
[illegible]
[illegible]
[illegible] may be [illegible]
or mixture of thyroid & pituitary may be given & [illegible]
causes restoration of sight
In lesions of optic tract the loss of vision in
one side of the field of vision – hemianopia
[illegible] may occur in internal capsule
may be [illegible] loss of
vision of corresponding [illegible]

DISEASES OF THE OPTIC NERVE.

CONGENITAL ABNORMALITIES: Coloboma, opaque nerve fibres, "hole."

OPTIC NEURITIS: (*a*) Intra-ocular. (*b*) Retro-ocular.

(*a*) INTRA-OCULAR NEURITIS: Characterised by infiltration of the nerve-head with inflammatory exudation, and consequent swelling of the papilla and distension, tortuosity and some obscuration of the retinal vessels. When the swelling of the papilla is marked (+2D or more) and venous congestion pronounced, the condition is called papillœdema or choked disc; when œdema and venous congestion are less marked, it is called interstitial papillitis. When the exudation spreads into the surrounding retina, it is called neuro-retinitis. The disc appears enlarged, has a dull woolly reddish-grey appearance, and may be mottled with hæmorrhages, white spots or streaks. Persistent neuritis leads to atrophy of the nerve fibres with pallor of the disc and contraction of the vessels (post-neuritic atrophy).

Symptoms: Impairment of sight may or may not be complained of, and its presence is no indication of the severity of the disease. Attacks of temporary loss of vision sometimes. Defects of the visual field—peripheral restriction, dyschromatopsia, central scotoma, enlargement of blind spot, according to cause and type of neuritis. Gradual or sudden loss of vision, with atrophy of nerve. Associated symptoms referable to primary cause, *e.g.*: headache, vomiting, cerebral symptoms.

Causes: (i) Cerebral tumours—malignant, syphilitic, tuberculous, hæmorrhagic, thrombotic, &c.
(ii) Meningitis.
(iii) Orbital inflammation.
(iv) Toxæmia—Syphilis, tubercle, albuminuria, lead, exanthemata, nasal sinusitis.
(v) Heredity—Mainly retro-ocular neuritis.
(vi) Traumatism.
(vii) Lesions of spinal cord.

Treatment: General (*v.* causes). Decompression.

(*b*) Retro-Ocular Neuritis: An interstitial inflammation of the orbital portion of the optic nerve often affecting the macular fibres only, and showing little or no intra-ocular changes. It may be (*i*) Acute; (*ii*) Chronic.

(*i*) Acute Retro-Ocular Neuritis.

Causes: (i) Secondary to disease or injury of nasal sinuses or orbital structures.
(ii) Toxæmia: Rheumatism, infectious diseases, drugs.
(iii) Heredity (Leber).
(iv) Disseminated Sclerosis (recurrent).

Symptoms: Generally affects youth or adolescents. Failure of vision, more or less sudden, chiefly confined to central area (central scotoma), and preceded or accompanied by pain in and around orbit and on movement of and pressure on eyeball. Scotoma may be absolute or relative. Peripheral field may be restricted. Pupil reaction to light diminished or lost.

Prognosis: Good where cause can be removed (sinus disease, toxæmia, &c.). Hereditary cases seldom improve, but never become quite blind.

Treatment: Remove cause. Salicylates, diaphoretics, and blood-letting in toxæmia. Strychnine.

(*ii*) Chronic Retro-Ocular Neuritis ("Toxic Amblyopia").

Causes: (i) Tobacco.
(ii) Other poisons—Alcohol, lead, arsenic, carbon bisulphide, &c.
(iii) Vascular sclerosis.

Symptoms: Affects people in middle or later life. Vision gradually fails, the patient being impressed by his:—
(*a*) Inability to see as well in a bright light as in dull light = Nyctalopia or day-blindness.
(*b*) Inability to distinguish small coloured objects = Central Scotoma for colours.

(*c*) Impairment of vision both for distance and near = Amblyopia.

The disease affects both eyes and in more advanced stages the optic discs show temporal pallor. The condition is amenable to treatment if the supply of toxin is stopped.

Treatment: Remove the cause. Strychnine, potassium iodide, water.

ATROPHY OF THE OPTIC NERVE: Degeneration and shrinking of the nerve fibres, evidenced by a white or grey appearance of the optic disc. It may be:—

(a) Primary: Cerebro-spinal diseases, Heredity.

(b) Secondary: To inflammatory diseases of the optic nerve (consecutive or postneuritic), choroid or retina, retinal vascular obstruction, injury or compression of the nerve.

Characters:

Primary: Disc greyish-white, edges sharply defined; lamina cribrosa conspicuous; vessels slightly or not contracted.

Secondary; (a) Postneuritic: Disc white, filled in, and woolly, lamina not visible, edges ill-defined, vessels contracted, often white lined. (b) Retinitic: Disc greyish, or dirty red, vessels very thin. (c) Thrombotic: Disc very white, vessels thread-like.

Symptoms: Gradual failure of sight, with contraction of the visual field, or central scotoma. Impairment of colour perception.

Prognosis: Generally bad visually. Vitally favourable in tabes.

Treatment: General and constitutional. Strychnine. Decompression in neuritic stage.

TUMOURS OF OPTIC NERVE: Neuro-fibroma, endothelioma, glioma, sarcoma, myxoma, tubercle.

Symptoms: Impairment of sight. Proptosis, with little or no interference with ocular movements.

Treatment: Removal of growth by orbital resection or exenteratio.

CHIASMAL LESIONS.

Causes: Pituitary growths, distension of third ventricle, sphenoidal disease, injury.

Symptoms: Failing and variable vision. Bitemporal hemianopia with symptoms indicating increased intracranial pressure. Enlarged sella turcica (X-rays). Optic neuritis occasionally, generally atrophy (descending). Acromegaly, Infantilism, Frôhlich's Syndrome.

TRACT LESIONS: Homonymous hemianopia, with loss of pupil reflex. Wernicke's hemiopic pupillary reaction.

CORTICAL LESIONS: Homonymous hemianopia, with retained pupil reflex. Homonymous hemianopia contralateral to lesion. Wilbrand's prism test.

AMBLYOPIA, or impaired vision. Complete loss=amaurosis. Terms generally applied to defective vision without any visible organic lesion. It may be:—

(i) CONGENITAL: Commonly associated with high degrees of ametropia, or squint. (?) Amblyopia ex anopsia.

(ii) HYSTERICAL, OR NERVOUS: Generally unilateral in young girls, and associated with peripherally contracted or rapidly exhausted visual fields, inversion of the colour fields, anæsthesia of cornea, and other signs of hysteria, or neurasthenia.

(iii) HEMERALOPIA: Night blindness, congenital or acquired.

(iv) TRANSIENT AMBLYOPIA: Due to retinal ischæmia, trauma, heart failure, arterial spasm, migraine.

(v) SIMULATED:

Tests: Prism, Diploscope, Diaphragm, Haselberg's, Snellen's, Roche's.

THE LENS.

DEVELOPMENT AND STRUCTURE: Nucleus, Cortex, Capsule, Poles, Equator. Suspensory apparatus.

GROWTH AND NUTRITION.

FUNCTION: Accommodation.

DISEASES OF THE LENS.

CATARACT, or Opacity of the Lens.

Classification:

(i) Congenital or infantile—partial (lamellar or zonular, anterior or posterior polar, cortical "dot," axial), or complete.

(ii) Idiopathic—"senile," nuclear, cortical, nucleo-cortical. Generally "hard."

(iii) Toxic—diabetes, naphthalin, etc., as (ii) but "soft" under 25.

(iv) Complicated or secondary—Uveal, or retinal disease, glaucoma. Often posterior cortical (bowl-shaped).

(v) Traumatic—irritation of capsule (acquired anterior polar v. Ophth. Neonatorum), perforation of capsule, concussion, intense light and heat.

(vi) After-cataract, also called secondary. Opacification of capsule after cataract extraction.

Objective features of Nuclear, cortical, nucleo-cortical lamellar, anterior and posterior polar, posterior cortical, hard and soft cataracts, after-cataract.

Symptoms: Dimness of vision $\frac{6}{6}$ to P.L., never V=O. Dazzling—dull light preferred. Polyopia, photopsia, muscæ, myopia and shallow A.C. from swelling of the lens. Glaucoma, if swelling is very rapid. Nystagmus in dense infantile cataract.

Stages of senile cataract:

(a) Incipient: Opacity slight; V. still useful.

(b) Immature or unripe: Iris casts shadow on lens; fundus reflex still visible.

(c) Mature or ripe: Opacity extends to capsule, iris casts no shadow, fundus reflex not visible.
V=hand movements.
A sclerosed cataract never becomes quite opaque, but remains translucent—"black" cataract.

(d) Hypermature: Liquefied (Morgagnian), shrivelled calcareous, capsule thickened.

Course: Congenital and infantile generally non-progressive; other forms progressive. Senile nuclear generally slow, cortical and nucleo-cortical quicker. Unwise to prophesy as to rate of development.

Operability: Maturity. Eye free from disease, especially of lachrymal and conjunctival sacs. Pupils active and equal. Projection of light good. Tn. Search for possible foci of infection or toxaemia (oral sepsis, &c.).

Treatment:

(a) Congenital, non-progressive:
If V. not less than $\frac{6}{18}$=nil.
If V. less than $\frac{6}{18}$=needling, or (?) iridectomy.

(b) Progressive:
Palliative. Dark glasses. Mydriatic. Iridectomy.
Operative: (i) Hard. Extraction through large limbic incision after preliminary iridectomy, with iridectomy (combined operation)—without iridectomy (simple extraction). (ii) Soft. In patients under 25 years of age, needling and removal of softened and loosened lens matter through a small corneal incision (evacuation). (iii) After-cataract. Tear or cut membrane with knife-needle (discission), or extract with forceps through small corneal incision.

After-treatment: Loss of lens (aphakia)=loss of refractive power, which must be made good by wearing strong convex glasses (+10 D for distance, +14 D for reading —prox.). There is generally some temporary astigmatism, and occasionally this is permanent and must be corrected.

DISLOCATION OF THE LENS (Ectopia lentis): Displacement, partial (sub-laxation) or complete (dislocation), of the lens from its normal position owing to absence or rupture of the suspensory ligament.

Ætiology: (i) Congenital—hereditary, bilateral.
(ii) Traumatic—blows, etc.
(iii) Degenerative—secondary.

Displacements may take place:

(a) Under conjunctiva—through rupture of sclera just behind margin of cornea.

(b) Into anterior chamber—golden-edged mass. Glaucoma.

(c) Into vitreous—laterally, vertically, or backwards. Free edge may be seen in pupil, iris tremulous (iridodonesis), monocular diplopia. Loss of accommodative power. Deepening of A.C. Glaucoma.

Treatment: Correct refractive and accommodative defects—spectacles. Extract lens from subconjunctival tissue and anterior chamber. Leave in vitreous. Excise eyeball, if blind and painful from glaucoma.

DISEASES AND INJURIES OF THE VITREOUS.

CONGENITAL ANOMALIES: Hyaloid artery, Cloquet's canal.

MUSCÆ VOLITANTES.

OPACITIES. Fine, flaky, massive, membranous, glistening golden or silvery (synchisis scintillans).

HYALITIS: Secondary to disease of contiguous structures, or traumatic septic injection (panophthalmitis).

HÆMORRHAGES: Injury or disease of choroid or retina. Recurrent hæmorrhages in young men. (?) Toxæmia. (?) Tuberculous.

FOREIGN BODIES: Perforating wound of globe.

Methods of localisation: Ophthalmoscope, X-rays, sideroscope, magnet. Field of vision.

Methods of extraction—of magnetic and non-magnetic. Forms of magnets and method of use. Dangers of retained F.B. Signs of retained steel: Siderosis, cyclitis, etc.

Treatment of Complications and after-effects v. Cyclitis, Cataract, etc.

Vitreous

Has no special blood supply in foetal life supplied by hyaloid artery through Cloquet's canal. Derives nourishment from vessels of choroid, retina & ciliary body. Fluid passes from behind forwards. No true inflammatory changes — secondary inflammatory invasion from surrounding structures. When such changes begin [illegible] vitreous has to expand, if seen does this normally, but if invaded with inflammatory exudate it contracts & may detach retina.

Injuries — foreign bodies which [illegible] & become [illegible] as there is some protective [illegible]

Sometimes vitreous becomes relatively fluid probably due to inflammation or morbid conditions in structures round. Small particles in vitreous not seen by ophthalmoscope — cause spec. to be seen. They do not do much harm. Sometimes diffuse opacities in vitreous — may [illegible] with vision. Sometimes dense cloud of dust-like opacities in vitreous & may cause great deal of [illegible] — [illegible] forwards — may disappear. [illegible] of choroidal [illegible]. May be haemorrhage into vitreous due to blow or occasionally [illegible] to [illegible]. [illegible] & [illegible] with sight gradually.

Abnormalities — Sometimes Cloquet's canal filled with pigment. Long black thread in [illegible] of vitreous.

GLAUCOMA.

Lymph channels of the eye.

Secretion and excretion of aqueous humour.

Anatomy of the corneo-iridic angle, canal of Schlemm and its venous connections.

Physiology of intra-ocular pressure.

GLAUCOMA is dependent on excess of intra-ocular pressure, and the symptoms are attributable to pressure effects on the various ocular structures.

Persistent excess of pressure depends on retention.

PRIMARY GLAUCOMA (not due to other diseases of the eye).

Active causes: Hyperæmia of the uveal tract, especially venous congestion—from fatigue, hunger, chill, shock, worry, grief, loss of sleep, constipation, cough, menopause.

Disease of choroidal and retinal vessels (especially thrombosis). Blood disorders. Nerve disorders.

Dilatation of the pupil by mydriatic—thickened iris base blocks up filtration angle.

Predisposing conditions: Advanced age—lens large, circumlental space small. Small eyeball—small cornea, hypermetropia. Shallow A.C. Female sex.

(a) Acute Primary Glaucoma (Congestive; pressure rises suddenly).

Symptoms and changes:

Transient obscurations of sight; rainbow rings round lights—from transient œdema of cornea. Accommodative failure—frequent change of reading glasses.

Ciliary injection, chiefly episcleral veins—from pressure on choroidal veins; may be intense with œdema simulating inflammation.

Anterior chamber shallow—lens pushed forwards.

Pupil dilated and iris discoloured from compression of iris base.

Cornea hazy and anæsthetic from subepithelial œdema and pressure on ciliary nerves.

Pain from stretching and compression; may be intense, causing vomiting and prostration—"bilious attack."

Tension increased (T+1, T+2, T+3).

Rapid failure of V. from obstructed circulation.

Blindness, with persistent pain and tension, or gradual shrinking of globe.

(b) Chronic Primary Glaucoma (Non-congestive, pressure rises gradually).

No premonitory obscurations and rainbows.

No ciliary injection in early stage or chief veins only.

No dilatation of the pupil.

No haze of cornea. No pain.

Tension increased at first slightly, later more so.

Gradual failure of V. Fields contracted, especially nasally (step).

Absolute or relative, central scotoma continuous with blind spot (Bjerrum's sign).

Cupping of optic disc: Lamina cribrosa yields to pressure, and nerve fibres become atrophied.

Pulsation of retinal arteries.

Chronic may pass on to acute form. Danger of mydriatics after middle age.

Diagnosis: Acute Glaucoma from iritis and scleritis. Chronic Glaucoma from cataract, and toxic amblyopia.

Treatment: Objects—to open filtration angle, and failing that to set up an artificial communication between the anterior chamber and subconjunctival space. To lower blood pressure; to prevent recurrence of uveal and retinal congestion.

Miotics—Eserin pilocarpin (cocain with miotics only).

Rest, warmth, sleep, morphia, blood-letting, purgation.

Iridectomy (acute). Sclerectomy, with or without iridectomy. Sclerotomy—posterior, anterior.

SECONDARY GLAUCOMA. (Complicating other eye diseases.)

Causes (leading to retention):

(1) Posterior synechiæ, with blocking of pupil.
(2) Anterior synechiæ—wounds, ulcers, operation.
(3) Swollen (injured or diseased) or dislocated lens.
(4) Intraocular tumours.
(5) Intraocular hæmorrhages.
(6) Cyclitis, with albuminous exudation.
(7) Congenital malformations; aniridia, buphthalmos.

Symptoms: Those of primary disease, with those of primary Glaucoma superadded.

Treatment: Deal with cause—iridectomy, removal of lens, tumours, etc., subconjunctival injections (Sod: citrat:). Excision.

BUPHTHALMOS: Congenital Glaucoma, or ox eye. Often hereditary and bilateral. Due to increased intraocular pressure in early life. Whole eye, including cornea, becomes distended and enlarged with cupping of O.D. Kerato-globus is a form of infantile glaucoma.

Causes: Congenital absence of canal of Schlemm. Imperfect differentiation of iris and cornea.

Treatment: Miotics. Iridectomy (?) Sclerotomy. Sclerectomy. Excision.

REFRACTION. VISUAL ACUITY. ACCOMMODATION.

REFRACTION means the optical condition of the eye when not accommodating, and refers to the position of the principal focus (image of distant object) in relation to the retina.

Forms:

(i) Emmetropia (E.) distant object pictured on retina.

(ii) Ametropia, distant object not pictured on retina.

(a) Hypermetropia (H.) distant object pictured behind retina.

(b) Myopia (M.) distant object pictured in front of retina.

(c) Astigmatism (As). Perfect picture impossible. Refraction differs in different meridians.

(iii) Anisometropia. Difference in the refractive error of the two eyes.

Methods of estimating refraction:

(i) Subjective method.

(ii) Objective methods:

(a) Retinoscopy.

(b) Ophthalmoscopy.

(c) Ophthalmometry (Keratometry).

(i) Subjective method: Use of test lenses. Meaning of dioptre, + and −, spherical, cylindrical, and toric lenses. Numbering of lens, notation of axes of cylinders. Measurement of lenses. Principle of Snellen's test types. Astigmatic fan. Select strongest convex and weakest concave that gives best V. Test for astigmatism with minus or crossed cylinders. Test both eyes separately and together.

(ii) Retinoscopy. Skiascopy or shadow test. Consists in the observation of the light reflex from the fundus and the shadows which surround it when light is reflected into the eye from a retinoscope (a flat mirror with a hole in it).

Hold retinoscope at fixed distance (arm's length). When it is rocked from side to side or up and down shadows will invade the red-lighted pupil from either side, and their movements indicate the refraction of the eye :—

Emmetropia—move with mirror (relative H. for distance of observer).

Hypermetropia—move with mirror.

Myopia—move against mirror.

An emmetrope made artificially myopic with a +1D lens shows no shadow at 1 metre (arm's length).

Lenses are put up before the eye tested until the shadows disappear in all meridians; the estimate is then corrected for the distance of observation (1 metre) by adding −1D to the shadow-obliterating lenses. If a cycloplegic has been used another −·50D or −1D is added.

Use cycloplegic in

(a) Myopes under 20 years of age.

(b) Young squinters.

(c) Young astigmats.

(d) Hypermetropes under 20, and under 40 if asthenopia persistent after correction without cycloplegic.

(e) Very small pupils or indistinct shadows.

ERRORS OF REFRACTION.

HYPERMETROPIA: Long Sight Parallel rays focussed (virtually) behind the retina. Eye too short ("flattened") for its focal length. All or part of it may be hidden by involuntary accommodation=*latent* H., the remainder is called *manifest* H., the two together constitute *total* H.

Total H. shown under cycloplegic, and difference between this and H. discovered by subjective testing= latent H.

Causes:

(1) Imperfect growth of eyeball—axis too short.
(2) Abnormalities of cornea and lens.
(3) Aphakia.
(4) Senility.

Symptoms: Strain of accommodation—fatigue, pain in eyes and head (Asthenopia).

Failure of accommodation—dim V., especially in near V.

Associated convergence excess—squint or Esophoria, Hyperæmia of lids, conjunctiva, etc.

Treatment: In young—often sufficient to correct manifest H. only. Generally correct half latent H. as well. In middle age and squinters correct total H. In many cases glasses only necessary for near V.

MYOPIA: Short sight. Parallel rays focussed in front of retina, the eyeball being too long for its focal length.

Causes:

(1) Elongation of the globe—from hereditary predisposition, debility, malnutrition, excess of close work (convergence and stooping).
(2) Abnormal suspension or tilting of lens—traumatic or congenital luxation).
(3) Densification of nucleus (myopic degeneration).
(4) Abnormalities of cornea—keratoconus, etc.
(5) Spasm of accommodation.

Myopia rare in infancy. Generally noticed between 8 and 12. Educational period 12 to 18 most dangerous for increase (prolonged convergence, stooping, vitiated atmosphere). Rarer in country than in towns, in lower than higher schools and universities, in lower classes of society than in higher.

Symptoms and changes: Gradual failure of distant V., getting worse during period of growth. Thinning of sclera at posterior pole; in high degrees, tears, hæmorrhages at macula, detachment of retina, divergent strabismus.

Prevention and treatment: Avoid close work in early life. Regulate sitting position, light, work and hours. Open air exercise and work, if possible. No book work when fatigued or badly fed. Oral education only. Correct refraction fully and periodically.

ASTIGMATISM: One meridian varies from another in refractive power. Rays proceeding from a point are not focussed in a point. The defect is mainly in the anterior surface of the cornea (*v.* Keratometry), but may be largely lenticular. It is best estimated by retinoscopy.

Irregular Astigmatism is due to unsystematic irregularities in the surface of the cornea or in the structure of the the lens.

Regular Astigmatism. The extremes of curvature are at right angles to each other; the axis of the astigmatism as that of the correcting cylinder being that which is nearer the Emmetropic. The commonest relation is for the vertical meridian to possess the greater, and the horizontal the less, curvature. This is called astigmatism "with the rule," and the converse is called perverse astigmatism, or As. "against the rule." The latter gives rise to more asthenopia than the former.

Varieties: Simple H. As. Simple M. As. Compound H. As. Compound M. As. Mixed astigmatism.

ERRORS OF ACCOMMODATION.

PRESBYOPIA, or old sight. Corrected by wearing convex or weaker concave lenses. A normal condition. Begins 45 to 50 years of age when P. recedes beyond convenient reading distance (15 inches) as evidenced by pushing away of reading matter, throwing back head, reading facing light to contract pupil and sharpen image. Early presbyopia sign of hypermetropia, and also of glaucoma. Late onset in myopia. Accommodation is abolished in aphakia and dislocation of lens. Debility of accommodation occurs in neurasthenia, and after illnesses, loss of sleep, parturition, lactation, etc.

PARALYSIS AND PARESIS of accommodation:

(1) Use of cycloplegics (atropine, etc.).
(2) Toxæmia: Diphtheria, diabetes, syphilis, etc.
(3) Parasyphilis. Tabes, G.P.I.
(4) Contusions.
(5) Glaucoma (paresis).

Treatment: Remove cause, if possible. Constitutional. Tonics. Pilocarpin (stimulant of ciliary muscle). Convex lens, if symptoms persistent.

SPASM OF ACCOMMODATION

(1) In young myopes, and sometimes in older people. (Refraction altered under cycloplegic.)
(2) Hysteria (with excessive convergence).
(3) Miotics.

Treatment: Cycloplegics. Nerve sedatives.

ERRORS OF MOTILITY.

(i) Muscular Imbalance; Heterophoria, or latent squint.
(ii) Strabismus, or squint.
(iii) Muscular Paralysis.

(i) **HETEROPHORIA**: Abnormal ocular muscular balance.
[Orthophoria=normal ocular muscular balance.]

Heterophoria may be
(1) Esophoria—latent convergence.
(2) Exophoria—latent divergence.
(3) Hyperphoria—latent vertical deviation.

Causes: Impaired health, insufficient innervation, malposition of muscles.

Symptoms: Pains in eyes and head; difficulty in reading, etc.; occasional diplopia, dizziness or giddiness (Asthenopia).

Tests: Maddox rod—tendency to fusion removed by altering character of one image by means of a glass rod. Eyes then assume latent tendencies. Degree of deviation measured by prism required to bring images together or by tangent scale. Fusion power measured by prisms overcome. Normally:—
Internal Recti = 20°—30° (P. base out).
External Recti = 7°—8° (P. base in).
Superior and Inferior Recti = 3°—4° (P. base up or down).

Treatment: Constitutional and tonic. Correct errors of refraction. Prism exercises for weak muscle. Prism to ease weak muscle—about half the error being corrected. Operative: Tenotomy of overstrong, advancement of weak muscle.

(ii) **STRABISMUS**, or Manifest Squint: Both lines of sight are not directed towards the same object of fixation.

Varieties:

Convergent S.=one eye turned in.

Divergent S.=one eye turned out.

Sursumvergent S.=one eye turned up.

Deorsumvergent S.=one eye turned down.

Occasional or periodic S.=latent between the periods.

Alternating S.=affecting sometimes one eye, sometimes the other.

Concomitant S.=the angle of deviation between the two lines of sight remains the same for any point in the field of vision equally distant from the eye.

Paralytic S.=when concomitancy does not hold.

Measurement of S. angle, or amount of Squint.

(a) By Priestley Smith's tape.
(b) By Perimeter.
(c) By position of mirror reflex on cornea.

Apparent from real Squint diagnosed by exclusion test and by position of corneal reflex of mirror.

CONVERGENT STRABISMUS (Concomitant).

Ætiology: Appears generally between the ages of 2–5 years, being occasional at first, later constant.

(1) Hypermetropia and astigmatism.
(2) Amblyopia in one eye (congenital, ex anopsia, or medial opacities).
(3) Hereditary tendencies.

Theories of mode of production:

(1) Developmental inequality, or faulty insertion of muscles.
(2) Accommodative theory.
(3) Failure in development of fusion sense.

Symptoms: Deviation. Primary being equal to secondary.

Movements good in all directions.

Loss of binocular vision—no diplopia (*cf.* Paralytic).

Varieties of squint vision:

(1) Suppression of one image.
(2) Simultaneous vision.
(3) Superposition of images.
(4) Fusion, with stereoscopic V.

Tests for binocular vision.

Treatment: Correct errors of refraction.

Education of amblyopic eye by covering, or atropinisation of good eye.

Education of fusion sense by amblyoscope, stereoscope, diploscope, etc. (orthoptic exercises).

Readjustment of muscles by operations, viz.:

10° –15° Tenotomy of Int. Rectus or advancement of External.

20°—25° Tenotomy of Int. Rectus with advancement of Ext. Rectus.

30°—60° Tenotomy of one or both Internal Recti, advancement of External.

DIVERGENT CONCOMITANT STRABISMUS.

Causes: May appear at any age. Often associated with Myopia, or loss of V. in one eye. Post operative (Internal tenotomy).

Treatment: Correct refraction. Advancement of Internal Rectus, or External tenotomy, with or without advancement of Internal, followed by orthoptic exercises.

MUSCULAR PARALYSES: May be classified according to the nerve or muscle involved.

INTERNAL ophthalmoplegia—sphincter and ciliary muscles paralysed.

EXTERNAL ophthalmoplegia—all extrinsic muscles paralysed.

TOTAL ophthalmoplegia—all extrinsic and intrinsic muscles paralysed.

Causes: Congenital (often with levator palp.). Syphilis, Tubercle, Rheumatism and Gout (Toxæmia). Trauma. Fracture of skull, cerebral tumours, meningitis, periostitis.

Symptoms: Diplopia. Confusion of V. Dizziness or giddiness. Limitation of movements of the globe. Head held away from paralysed side to avoid diplopia. Secondary deviation (of sound eye) excessive. False projection and orientation.

Diagnosis of muscle affected :

(1) The false image appears to the patient to occupy a position relative to the true which represents the physiological action of the muscle paralysed.

(2) Whichever movement separates the images most, the paralysed muscle is concerned in that movement.

(3) The inefficient muscle belongs to the eye whose image stands farthest in the direction of the movement.

Treatment: Causal. Potass. Iod., Sod. Salicylat., Diet, etc. Occlude eye to remove diplopia. Exercise, galvanisation, shortening of paralysed muscle.

NYSTAGMUS: An involuntary oscillatory movement of the eyeballs. The movement may be from side to side (lateral N.), up and down (vertical), round antero-posterior axis (rotatory), or a combination of these (circumductory).

Ætiology: Congenital defects—lack of choroidal pigment (albinism) and other malformations with defective V.
Acquired visual defects—opacities of the media.
Diseases of the nervous system —disseminated sclerosis, cerebellar disease, etc. Disease or injury of labyrinth.

COAL MINER'S NYSTAGMUS: Found among miners who work in badly lighted coal pits. The nystagmus is apparently due to the inability of the fovea of the dark-adapted eye to see and fix small objects (coal facets) for any length of time, and the search for more defined vision in the perifoveal region, which has no point of commanding sensitiveness to maintain fixation. Often associated with photophobia, headache, giddiness, night-blindness, and spasm of lids, etc.

Nervous instability, errors of refraction, injury, disease, alcoholism, bad ventilation, act as contributory factors.

Treatment: Change of occupation. Remove contributory causes, if possible. Nerve sedatives, Tonics, Miotics.

ORBIT.

Boundaries and contents of orbit.

PERIOSTITIS.

Causes: Trauma, tubercle, syphilis, extension from neighbouring sinuses.

Symptoms: Pain, tenderness, swelling, perhaps abscess, fistula, and later ectropion.

Treatment: Constitutional. Hot fomentations. Incision.

ORBITAL CELLULITIS. Inflammation of the cellular tissue of the orbit, usually terminating in suppuration.

Causes: Trauma (often undiscovered F.B.), Erysipelas, Septicæmia, necrosis of walls, extension from sinuses.

Symptoms: Constitutional disturbance, œdema and redness of lids, chemosis, proptosis, loss of motility of eye, perhaps panophthalmitis and meningitis. V. may fail from optic atrophy due to venous thrombosis or cicatricial pressure.

Danger: Cavernous sinus infection and meningitis.

Treatment: Hot fomentations. Early incision.

TUMOURS: May arise in orbit, or spread from neighbouring nasal sinuses, or be metastatic. The following may be met with:—Cysts, aneurism, angioma, osteoma, endothelioma, sarcoma, carcinoma. (*v.* also tumours of optic nerve and lachrymal gland.)

Symptoms: Displacement of eye, with limitation of its movements from mechanical interference, infiltration, or nerve paralysis.

Tumours may be palpable, with pulsation in true and arterio-venous aneurisms, varying on stooping, etc., in angioma.

Treatment: Excision of primary growths through incision of lid (orbitotomy), after resection of outer wall of orbit (Krönlein's operation), or after removal of the eyeball (exenteratio).

EXOPHTHALMOS or PROPTOSIS: May be due to

(1) Orbital cellulitis or periostitis.
(2) Thrombosis of cavernous sinus.
(3) Suppuration or tumours of nasal sinuses.
(4) Foreign body in orbit.
(5) Tumours in orbit.
(6) Hæmorrhage or emphysema.
(7) Exophthalmic goitre.
(8) Complete paralysis of ocular muscles.

ENOPHTHALMOS or RECESSION OF THE EYEBALL: May be due to

(1) Emaciation.
(2) Non-penetrating injuries, with or without (?) fracture of orbital walls.
(3) Cicatrices after injury and cellulitis.
(4) Paralysis of the sympathetic (with miosis and ptosis= Horner's S.)
(5) Maldevelopment of ocular muscles.

INJURIES OF THE ORBIT.

(a) Non-penetrating. Blows, etc.
(b) Penetrating wounds.

(a) Orbital hæmorrhage, proptosis, fracture of wall. Optic nerve injury with descending atrophy and partial or complete loss of V. from fracture of optic ring, hæmorrhage into nerve sheath, concussion of nerve or callus formation.
Emphysema of lids when air sinus opened.
Secondary complications—periostitis, necrosis, abscess, meningitis.

(b) Injury of orbital contents, walls and even brain.
Gunshot injuries—direct and indirect effects on orbital structures, eyeball and optic nerve.
Foreign bodies may be retained unsuspected in orbit.

Treatment: Open up wounds and render as aseptic as possible. F.B.'s should be removed if causing irritation and are accessible. Localised by X-rays. In case of fracture of walls, treat as fracture of skull.

LACHRYMAL APPARATUS.

Structure and functions of:—

(1) Lachrymal gland and its ducts.
(2) Lachrymal canals.
    (a) Puncta and canaliculi.
    (b) Lachrymal sac.
    (c) Nasal duct.

DISEASES OF THE LACHRYMAL GLAND.

DACRYOADENITIS.

Acute: In children with purulent conjunctivitis. Abscess may result.

Chronic: Simple, tubercular, metastatic.

When associated with chronic hyperplastic inflammation of salivary glands=Mikulicz's Disease.

TUMOURS OF THE GLAND.

(a) Cystic (Dacryops) of gland or ducts.
(b) Tubercle, syphilis.
(c) Adenoma, fibroma, endothelioma.
(d) Sarcoma, carcinoma.

DISEASES OF THE LACHRYMAL CANALS.

Lachrymation is the hypersecretion of tears. Epiphora is the overflow of tears due to impaired outflow, or hypersecretion from reflex irritation.

PUNCTA: Congenital absence, displacement (ectropion, viith N. paralysis). Stenosis.

CANALICULI: Abscess, Concretions (streptothrix, &c.), F.B., stricture, pressure from without.

NASAL DUCT: Epithelial plug (congenital), stricture, inflammation of mucous and submucous tissue, or bony canal. Pressure of tumours of adjacent tissues.

DACRYOCYSTITIS:

(a) Acute—suppurative.
(b) Chronic—catarrhal or granulomatous.

(a) ACUTE: Lachrymal abscess, also involving surrounding tissues, due to secondary infection of (b) with virulent pyogenic organisms.

Symptoms: Acute pain and tenderness, redness, œdema in sac region (*cf.* erysipelas). Often pus regurgitates through canaliculi on pressure of distended sac. Abscess may burst and lead to fistula.

Treatment: Prophylactic: Treat (b). Hot fomentations, incision and drainage. Treat obstruction in duct only when all acute inflammation has subsided. Fistula: Excise track and sac.

(b) Chronic Dacryocystitis: Generally follows obstruction of duct.

(i) Catarrhal, generally pneumococcal.
(ii) Tuberculous.
(iii) Syphilitic.
(iv) Trachomatous.
(v) Periostitis or caries of bony wall (syphilis, trauma).
(vi) Tumours of adjacent structures—nose, jaw.

Primary infection often nasal, and nose should be examined in all cases for rhinitis, sinusitis, etc.

Symptoms: Epiphora ("watery eye"), conjunctivitis (lachrymal), and associated lid troubles. Distension of lachrymal sac with mucoid or mucopurulent discharge (mucocele), which can generally be made to regurgitate along the canaliculi by pressure over saccular swelling.

Dangers of chronic dacryocystitis: Acute suppuration (a). Infection of corneal wounds=Serpiginous ulcer with hypopyon, post-operative suppuration.

Diagnosis. From empyema of frontal and ethmoidal cells, and orbital pus pointing at inner canthus.

Treatment: Palliative=Instillation of antiseptic and astringent drops (Zn. Ag., etc.) immediately after emptying sac by pressure. Treat nasal disease. Syringing of sac with astringents and antiseptics. Probing of nasal duct after dilatation of canaliculi, and leaving styles in for indefinite period. In resistent cases excision of sac gives very satisfactory results

Excision of the lachrymal sac—methods.

EYE SYMPTOMS IN SYSTEMIC DISEASES.

NERVOUS SYSTEM.

INTRACRANIAL TUMOUR:

Optic neuritis; papillœdema; optic atrophy.
Retinal degeneration and hæmorrhages.
Paralysis of ocular muscles.
Nystagmus.
Intermittent amblyopia; permanent failure of central V., often late.
Disturbances of the visual field—hemianopia, irregular contraction, scotoma, dyschromatopsia.

TABES DORSALIS AND G.P.I.:

Reflex iridoplegia—"Argyll Robertson Pupil."
Miosis.
Paralysis of extrinsic muscles.
Optic atrophy—grey, primary.
Field of vision—peripheral contraction, central scotoma, dyschromatopsia.

DISSEMINATED SCLEROSIS:

Retrobulbar neuritis—central scotoma; temporal pallor of O.D.; optic atrophy.
Nystagmus.
Ocular paralyses.
Anisocoria.

MENINGITIS:

Paralysis of ocular muscles.
Nystagmus.
Optic neuritis.
Choroidal tubercles (miliary T.)

CEREBRAL HÆMORRHAGE, THROMBOSIS & EMBOLISM:

Homonymous hemianopia; conjugate deviation of the eyes.

HYSTERIA:

Anæsthesia of cornea, conjunctiva and lids.

Spasm or paresis of ocular muscles—ptosis, etc., cycloplegia, etc.

Amblyopia or Amaurosis.

Disturbances of visual field: contraction (tubular), scotoma, dyschromatopsia.

Diplopia or Polyopia.

Photophobia.

NEURASTHENIA:

Accommodative and retinal asthenopia.

Rapidly increasing contraction of the field of V. (exhaustion spiral).

Twitching of lids and inability to close them completely in "Romberg position."

DISEASES OF THE CIRCULATORY SYSTEM.

ANGIO-SCLEROSIS:

Recurrent subconjunctival hæmorrhages.

Thrombosis of retinal arteries and veins.

Tortuosity, kinking, dilatations of vessels.

Exaggerated reflex ("Silver-wire").

White lining of arteries.

Cataract, glaucoma, central scotoma.

CHOROIDAL SCLEROSIS: "Tigroid fundus."

Secondary pigmentary deposits.

Colloid degeneration of lamina vitrea.

ENDOCARDITIS:

Embolism of retinal artery.

Retinal hæmorrhages.

Pulsation of retinal arteries (aortic regurgitation).

ANÆMIA AND BLOOD DYSCRASIAS:

Dilatation and flattening of veins.

Retinal hæmorrhages and degenerative patches.

Optic neuritis and atrophy.

Amblyopia.

Accommodative and retinal asthenopia.

DISEASES OF THE URINARY SYSTEM.

BRIGHT'S DISEASE:

Hæmorrhages and degenerative changes (starfigure, etc.) in the retina.

Neuro-Retinitis with exudation.

Uræmic amaurosis.

Œdema of the lids.

Subconjunctival haemorrhages. Iritis.

DIABETES:

Cataract and transient myopia.

Iritis, cyclitis, vitreous opacities.

Hæmorrhagic retinitis.

Retrobulbar neuritis.

Paresis of accommodation, transient hypermetropia and premature presbyopia.

DISEASES OF THE DUCTLESS GLANDS.

GRAVES'S DISEASE:

Exophthalmos. Partial anæsthesia of cornea. Ulceration in advanced cases.

Eyelids: Retraction (Dalrymple), lagging (Graefe), infrequent nictitation (Stellwag), trembling when closed (Rosenbach), difficult of eversion (Gifford), pigmentation, transient œdema, tremor.

Epiphora.

Insufficiency of extraocular muscles (Mœbius).

Inequality of the pupils.

Pulsation of retinal arteries (Becker).

MYXŒDEMA AND CRETINISM:

Œdema and drooping of lids, tonic contraction of frontalis.

Lachrymation.

Optic neuritis and atrophy. Bitemporal hemianopia.

ACROMEGALY, INFANTILISM, &c. (PITUITARY GROWTHS):

Bitemporal hemianopia. Central Scotoma.

Optic atrophy and neuritis.

Oculomotor paralyses.

Nystagmus.

NASAL DISEASES.

RHINITIS:

Conjunctivitis, Keratitis (Phlyctenular, &c.)
Lachrymal obstruction and dacryocystitis.
Reflex asthenopia and photophobia.

ACCESSORY SINUS DISEASE:

Œdema of lids and conjunctiva.
Tenderness on pressure and movement of globe.
Recurrent episcleritis. Persistent asthenopia. Vertigo.
Retrobulbar neuritis, central scotoma, enlarged blind spot.
Paralysis of extrinsic muscles.
Displacement of globe and restriction of movements.
Palpable or visible tumour—inner canthus generally.
Orbital cellulitis.

INFECTIOUS DISEASES.

DIPHTHERIA:

Paralysis of ciliary or extraocular muscles.
Optic neuritis.
Direct infection of conjunctiva, cornea, &c.

MEASLES AND SCARLET FEVER:

Photophobia, conjunctivitis, blepharitis, dacryocystitis, corneal ulceration.

SMALLPOX:

Corneal ulcers (from pustules).
Palpebral abscesses, Dacryocystitis.

TYPHOID FEVER:

Conjunctivitis, Keratitis.
Optic Neuritis. Embolism and thrombosis of retinal vessels. Uveitis.

INFLUENZA:

Conjunctivitis, Keratitis.
Paresis of intrinsic and extrinsic muscles.
Optic neuritis.

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